

905 Angioplasty: Physiologic Measures

Sunday, March 16, 1997, 5:00 p.m.-7:00 p.m.
 Anaheim Convention Center, Hall E
 Presentation Hour: 5:00 p.m.-7:00 p.m.

905-19 Low Cardiac Event Rate in Patients With Deferred Angioplasty Due to Normal Coronary Flow Reserve

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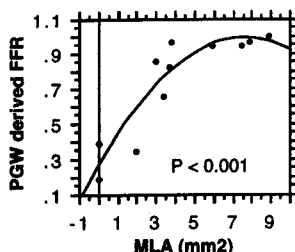
There is need for functional tests validating the hemodynamic significance of a coronary artery stenosis in the catheter laboratory. It was the objective of our study to determine the long-term cardiac event rate of deferring angioplasty in patients (pts) with lesions of angiographic significance however, normal coronary flow reserve (CFR). Our study included 69 consecutive patients with intermediate coronary artery stenoses (12 f, 57 m; diameter stenosis >40%, <90%) and an indication for PTCA due to stable angina pectoris and/or detection of silent ischemia in noninvasive stress test. CFR was measured distal to the lesion after intracoronary administration of papaverin or adenosin using 0.014 inch Doppler-tipped guide wire (FloWire™, Cardiometrics). In 23 pts (33%) PTCA was deferred (group A) due to CFR ≥ 2.0 . In the remaining 46 pts (67%) mean CFR of 1.4 ± 0.2 was measured (group B). CFR increased to 1.96 ± 0.5 after angioplasty. During follow-up (average 10 months) a re-PTCA was performed in group B in 6 pts (13%) because of unstable angina, while in group A only 1 angioplasty (4%) was performed in the vessel of interest. We did not observe any myocardial infarction (MI) in group A. In group B 3 pts (7%) had an acute MI.

We conclude that determination of the CFR is a valuable parameter for stratifying the hemodynamic significance of coronary artery stenosis. PTCA can safely be deferred in pts with significant coronary stenosis but a CFR ≥ 2.0 .

905-20 Validation of a New Guidewire for Measurement of Intracoronary Pressures and Fractional Flow Reserve

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Measurement of intracoronary pressure and the derived fractional flow reserve (FFR; distal/proximal arterial pressure at hyperemia) provides an accurate assessment of functional stenosis severity. We performed initial validation testing of a new 0.014" steerable, flexible-tipped pressure guidewire (PGW) with a fluid-filled lumen for pressure recording (Scimed/Boston Scientific). The PGW is easily connected to a small, disposable pressure transducer and standard cath. lab. monitoring system. *In-vitro* validation of pressure fidelity was comparable to standard guide catheters in frequency response (6 db power point = 45 Hz) and superior in critical damping. *In-vivo* testing was performed in 3 canines using a variable cuff coronary occluder producing a total of 11 stenoses, quantified with IVUS. Within the guiding catheter, phasic pressures from the PGW were super-imposed on the guide pressure. Correlation with a solid-state transducer was excellent in all positions in the artery ($r = 1.0$, $p < 0.0001$). A close relationship was shown between FFR and stenosis severity in terms of both % stenosis and minimum lumen area (MLA):



Conclusion: This new pressure guidewire offers a simple and inexpensive way to measure FFR, an accurate index of functional lesion severity.

905-21 Use of the PTCA Guidewire to Measure Artery Length and the TIMI Frame Count to Measure Time as a method of Calculating Coronary Velocity (= Length/Time) & Coronary Blood Flow

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The TIMI Frame Count (no. of frames for dye to first opacity standardized distal landmarks) is a relative index of coronary flow that measures the time for dye to reach the TIMI landmark. We describe here a method to measure the length to the TIMI landmark so that absolute velocity (length/time) can be calculated in pts. undergoing PTCA. At the end of a PTCA, the guidewire tip is placed at the TIMI landmark and a Kelly clamp is placed on the guidewire just as it exits the Y-adaptor. The guidewire tip is then withdrawn to the ostium of the guiding catheter and a second Kelly clamp is then placed on the wire just as it exits the Y-Adaptor. The distance between the 2 Kelly clamps outside the body is the same as the distance from the catheter ostium to the TIMI landmark. The velocity (cm/sec) = (length/frames) \times (frames filmed/sec). In 30 consecutive evaluable pts, the velocity \uparrow from 13.9 ± 8.5 pre-PTCA to 22.8 ± 9.3 cm/sec post PTCA ($p < 0.001$). Despite no change in TIMI Grade 3 Flow both before and after PTCA in 18 pts, the velocity actually \uparrow 38% from 17.0 ± 5.4 to 23.5 ± 9.0 cm/sec ($p = 0.01$). Flow (cc/sec) can be calculated by multiplying the velocity by the average cross-sectional lumen area along the length of the artery to the landmark. In the same 30 pts, the flow doubled from 0.6 ± 0.4 cc/sec pre-PTCA to 1.2 ± 0.6 cc/sec post-PTCA ($p < 0.001$). Again despite TIMI Grade 3 Flow both pre and post PTCA, coronary flow \uparrow 86% from 0.7 ± 0.3 to 1.3 ± 0.6 cc/sec ($p = 0.001$) ($n = 18$).

Conclusions: The distance to the TIMI landmark (length) can be simply measured using a PTCA guidewire. This length can be combined with the number of frames (time) to calculate measures of absolute velocity and flow that may be more sensitive to changes in perfusion than conventional TIMI Flow Grades.

905-22 Flow in Culprit Arteries Following PTCA is Faster Than That in Non-culprit Arteries: Timing & Magnitude of Hyperemia

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To determine the potential extent & magnitude of hyperemic flow detectable following PTCA, the post PTCA flow in culprit vessels was compared to post PTCA flow in nonculprit arteries & to normal reference values. Pooled data from pts. with acute coronary syndromes treated with either Tirofiban + heparin or heparin alone \times 36 hrs was analyzed. The no. of frames for dye to reach standardized distal landmarks was counted before & after PTCA to arrive at the Corrected TIMI Frame Count (CTFC). Before PTCA, flow was 34.4 ± 23.0 frames ($n = 1121$) which is 64% slower than the previously reported normal value of 21.0 ± 3.1 frames ($n = 78$) in pts. without acute ischemic syndromes ($p < 0.0001$). Post PTCA, the culprit flow was 14.7% faster than normal (hyperemic) (18.3 ± 10.4 , $n = 1165$ vs. 21.0 ± 3.1 , $p = 0.02$), & was also faster than nonculprits (23.6 ± 11.4 , $n = 417$, $p < 0.0001$). In a multivariable model controlling for catheter size and normal reference artery diameter, culprit flow was still faster than nonculprit flow ($p < 0.0001$). This observation was true in all 3 coronary arteries: LAD culprit (17.6 ± 10.7 , $n = 195$) vs LAD nonculprit (22.2 ± 10.8 , $n = 158$) ($p = 0.0001$); LCx culprit (20.3 ± 10.8 , $n = 310$) vs LCx nonculprit (25.0 ± 11.2 , $n = 227$) ($p < 0.0001$); & RCA culprit (17.2 ± 9.8 , $n = 605$) vs RCA nonculprit (20.8 ± 14.6 , $n = 32$) ($p = 0.05$). The 2nd injection was slower than the 1st, and flow returned to normal (18.6 ± 11.3 , $n = 608$ vs 21.2 ± 13.1 , $n = 102$, $p = 0.03$) with an associated 10% (2.6 frames) slowing in the 2nd injection of nonculprits.

Conclusions: Immediately following PTCA during the 1st injection, flow in culprit arteries is faster than previously reported normal values, and faster than nonculprit artery flow. By the 2nd injection, however, culprit flow appears to have returned to normal. Angiographic flow trials should control for the timing of injections after PTCA and nitrate administration.

905-23 Evaluation of Distal Coronary Flow During Angioplasty With Autoperfusion Balloons

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For protection of the myocardium during balloon angioplasty there are auto perfusion catheters (APC) available which are driven by the aortic blood pressure. It is suggested to use these devices in so-called high risk patients being endangered of hemodynamic collapse during balloon inflation. The quantity of the distal blood flow during balloon inflation in vivo is still unknown.